

Abstract

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Lipoprotein-associated phospholipase A2 activity is associated with coronary artery disease and markers of oxidative stress: a case-control study.

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BACKGROUND: Lipoprotein-associated phospholipase A(2) (Lp-PLA(2)) is a lipoprotein-bound enzyme that can release atherogenic isoprostanes from esterified phospholipids and that may be involved in inflammation and atherosclerosis.

OBJECTIVE: This study investigates the association between Lp-PLA(2) activity and coronary artery disease (CAD) in relation to oxidative stress markers, in particular urinary 8-epi-prostaglandin F(2alpha) (8-epi-PGF(2alpha)).

DESIGN: We conducted a case-control study in which the cross-sectional relation between Lp-PLA(2) activity, lipoproteins, and oxidative stress markers was determined in 799 patients with angiographically confirmed CAD and 925 healthy controls.

RESULTS: Lp-PLA(2) activity was significantly ($P < 0.001$) higher in CAD cases than in controls (32.9 ± 0.46 and 29.7 ± 0.42 nmol \cdot mL⁻¹ \cdot min⁻¹, respectively). Both elevated Lp-PLA(2) activity and urinary excretion concentrations of 8-epi-PGF(2alpha) were associated with greater CAD risk (P for trend < 0.001). Odds ratios for the upper quartiles of Lp-PLA(2) activity and 8-epi-PGF(2alpha) excretion were 2.47 (95% CI: 1.79, 3.40) and 2.19 (1.52, 3.15), respectively, after adjustment for sex, age, BMI, blood pressure, smoking and alcohol consumption status, and LDL and HDL cholesterol. When we examined the additive effect of both markers for CAD risk, the relation between 8-epi-PGF(2alpha) and CAD was weakened above the second quartile of Lp-PLA(2) activity. Moreover, Lp-PLA(2) activity was positively correlated with urinary excretion concentrations of 8-epi-PGF(2alpha) in controls ($r = 0.277$, $P < 0.001$) and cases ($r = 0.202$, $P < 0.001$) and with the tail moment of lymphocyte DNA ($r = 0.213$, $P < 0.001$) in controls.

CONCLUSION: This study shows an association of elevated Lp-PLA(2) activity with CAD risk in relation to oxidant stress and thus supports a proatherogenic role of Lp-PLA(2).

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